

Of more importance is the fibrosis accompanying chronic pericarditis or endocarditis. In these cases the myocardium becomes affected by direct extension or in consequence of circulatory disturbances. The terminal result of this condition, as in independent myofibrosis, must always be cardiac dilatation, if intercurrent disease have not prematurely terminated life. Before, however, this terminal condition has developed, clinical signs of failing circulation may arise as obscure indications of the myocardial weakness associated with the pericardial disease.

I shall not allude to the physical signs of simple pericardial disease, such as frictions, mammary or costal retraction, etc., but would recall the symptomatic and physical evidences of muscular incompetency in cases of combined pericardial and muscular disease. As in the independent myofibrosis, there may be a gradual loss of vigor, a premature senility, a tendency to irregularity of the heart to a peculiar grayish pallor, to gastric disturbances, etc., but all of these indications are less prominent than in the independent form because chronic pericarditis is more frequent at an early age, when the senile type of clinical manifestations are unlikely to occur.

The best evidences of myocardial association with pericarditis are therefore those derived from physical examination. First of all I wish to insist that marked hypertrophy of the heart develops in a surprisingly short time in acute cases. In a recent case in a boy of 9, I found such rapid enlargement of the heart that it seemed impossible that the enlargement could be other than dilatation. The autopsy, however, showed practically no dilatation and very great thickening of the walls. This enlargement, however, is not true hypertrophy; it is largely degenerative, and the clinical signs would indicate this. The pulse is weak and of low tension—out of all proportion to the degree of enlargement of the heart. The slapping, irritable impulse of Martius accompanied with a weakened and quick pulse is of prime importance in indicating myocardial association with the pericardial disease.

What I have just said refers especially to acute cases. The chronic cases are attended with less pronounced signs of myocardial disease, but it may be said with considerable emphasis that peripheral congestion or cyanosis, the peculiar enlargement of the liver that has been termed pericardial cirrhosis of the liver, the occurrence of dropsies and marked irregularities of the heart, are always indicative of associated myocardial trouble and therefore of grave prognostic significance.

I do not wish to be understood as saying that myocardial disease always accompanies pericarditis. In a strict pathologic sense there is probably always some involvement of the muscle of the heart, at least to the extent of slight sub-pericardial infiltration, but in a clinical sense the heart wall is often unaffected. It should, however, be recognized that even such slight myocardial disease may occasion striking signs and symptoms. Thus, Fisher has found great disturbance of the heart action and of the sounds—a Flint's murmur—when there was only a moderate sub-pericardial disease. In this connection I wish also to recall that Romberg has found in simple endocarditis thrombosis of the myocardial vessels and myocardial degeneration. This contribution is of very great importance, for in no other way could the marked disturbances of the heart's action be explained in cases of endocarditis with anatomically trivial lesions. I would couple this with what occurs

in pericarditis. The symptoms of the latter are largely the result of the underlying myocardial disease.

We are thus forced to the conclusion of Jürgensen that pancarditis is the diagnosis of the future, its type may be endocarditic, pericarditic, or myocarditic, but the immediate result and the final prognosis are to a large extent dependent upon the degree of involvement of the myocardium. A few dangers, such as general infection, embolism or mechanical interference with the heart by extensive effusion, are independent of the condition of the heart wall, but, these conditions excepted, the important criterion for prognosis is the condition of heart muscle.

ADHERENT PERICARDIUM.*

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It is so manifestly impossible in the few minutes allotted to me to consider this subject in its entirety, and so many of the points bearing on this subject have already been considered by preceding speakers, that it seems best to restrict my remarks to the clinical aspects of this disease.

We meet with adherent pericardium in two forms: 1. as a result of pericarditis interna which has led to a more or less complete and firm union of the two layers of the sac, without adhesion to the surrounding structures; 2, as a result of pericarditis interna et externa which has caused adhesion not only between the pericardium and epicardium, but also between the sac and the contiguous structures, as the chest wall, diaphragm and lungs.

In this second form there is often such an extensive development of fibrous tissue within the mediastinum, with consequent union of all the structures therein contained, that the condition has been termed chronic adhesive or fibrous mediastino-pericarditis. In some cases this proliferation of fibrous tissue is not limited to the mediastinum, but invades the pleural and peritoneal cavities in the form of a general serositis.

Not only is the capsule of the liver thickened, but the connective tissue hyperplasia invades the organ, becoming especially marked in the interior of the lobules and in the fissure. In time this fibrous tissue undergoes contraction and the liver becomes reduced in size and hard, very much as it does in hepatic cirrhosis. The once enlarged and chronically congested organ shrinks, becoming dense and thin bordered, more or less granular, but still fairly regular in outline, excepting that its notch is greatly exaggerated. According to Eisenmenger, it is by the contraction of the connective tissue within the fissure of the liver and consequent compression of the portal vessels that the shrinkage in the size of the organ leads to ascites, the same as does the atrophy in Laennec's hepatic cirrhosis.

Finally, the adherent pericardium may exist without chronic inflammatory changes in the endocardium or myocardium, but in most instances it is associated with valvular disease, or with chronic myocarditis.

SYMPTOMS.

These depend not only on the extent and situation of the pericardial adhesions, but also upon the co-existence, or not, of other lesions as valvular defects. If the two layers of the sac alone are adherent, and particu-

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larly if the adhesions are limited in extent, cardiac hypertrophy may be the only effect and the circulation is carried on so adequately that no symptoms are produced. If, on the contrary, the two surfaces of the pericardium become adherent, while the heart is in a state of dilatation from acute myocarditis, or in consequence of valvular disease, and if the organ is therefore not able to return to its previously small size, the effect upon the circulation is eventually disastrous and symptoms due to stasis declare themselves.

If the pericarditis was external as well as internal and led to fixation of the sac to some of the surrounding parts, the heart is held more or less firmly as in a vise and is hampered in its work. Its systoles are less effectual than normal, since the fixation of its walls serves to hold the cavities in a state of distension, and its contents are imperfectly discharged. Stasis inevitably results. If to this condition is united a valve lesion, as mitral regurgitation, the effect is particularly bad and symptoms are not long deferred. It is for this reason that chronic, adhesive mediastino-pericarditis is so likely to prove a serious affection. In all cases there is much difference in the severity and manifestation of symptoms. In one individual these are referable chiefly to stasis within the pulmonary vessels, shortness of breath on exertion, cough due to bronchial congestion and a marked disposition to attacks of bronchitis. In another case palpitations either with or without dyspnea are called forth by effort and excitement occasioning much discomfort and it may be alarm. In these cases the pulse is apt to be persistently rapid, it may be irregular and the cardiac impulse is exaggerated in force and extent. In a few cases without co-existing valvular defect, the disturbance of circulation is not pronounced, being shown only by digestive disorders lasting for years, and attributed to simple dyspepsia or to chronic gastritis, but rarely to their proper cause on account of the obscurity of the pericardial lesion and the difficulty or impossibility of its diagnosis.

In most cases there is nothing to distinguish them from ordinary instances of mitral disease or from cardiac dilatation. Physical examination usually discloses enlargement of the liver from passive congestion. This enlargement is generally greater than one would expect from the cardiac findings, and I have been impressed by the obstinacy of this hepatic engorgement under treatment. It is not only difficult to reduce its size by ordinary means of treatment, but the congestion of the organ displays a striking tendency to recur as soon as treatment is abandoned.

I have had under observation for several years a female patient whose mitral valve leaks and whose enormously enlarged heart is apparently completely encased in fibrous tissue that binds down the organ on all sides, so that no amount of rest in bed or digitalis seems to reduce its size in the least. The liver has always been greatly engorged, extending for a long time nearly to the crest of the ileum, and requiring the daily use of saline laxatives to relieve the patient from pain and discomfort. For the past year the organ has been gradually diminishing somewhat in size and growing in thinness and hardness. This patient has displayed remarkably little dyspnea on effort, but is greatly annoyed by the pounding and tumultuous action of the heart, this sensation being specially noticeable in the epigastrium. Of late she has had a great deal of cough, difficult mucous sputum and upon several occasions slight hemoptysis. She has to be extremely careful in her diet, and her urine and menses have become scanty.

Another female patient with pronounced mitral insufficiency has pericardial adhesions that bind down the left side and base of the heart, fixing the apex beat immovably in place far to the left and downward, but the border of the right heart is apparently free from adhesions. Whereas the left ventricle never varies in size under any conditions, the right heart, as shown by the area of cardiac dullness, becomes dilated with the greatest ease and rapidity. The liver, which is persistently enlarged, fluctuates somewhat in size in accordance with the state of the right heart, but even when at its smallest always extends from 2 to 3 inches below the inferior costal margin, no matter how vigorous may be the onslaughts upon it by means of epsom salts. This patient's symptoms are not of the digestive organs, but are those of shortness of breath and a rapid pounding action of the heart and general weakness. The urine remains fairly abundant and the menses are too profuse and protracted. She is always promptly benefited by absolute rest in bed, a milk diet, cathartics and digitalis, although this last never materially slows the heart.

When in all such cases compensation finally gives way the breakdown is complete and irreparable. Symptoms of stasis declare themselves everywhere, but do not differ materially from those of cardiac astylosism from any other cause.

The most interesting class of cases are those whose clinical features resemble a case of atrophic cirrhosis of the liver in its terminal stage. These are the cases of chronic fibrous mediastino-pericarditis. They not infrequently pursue a latent course for many years, and even after symptoms have set in are not recognized as pericardium until they come to autopsy. This applies particularly to cases in which the pericardial synechia is unassociated with valvular disease. Not only is there chronic engorgement of the liver, but the contraction of the fibrous tissue interferes at length with portal circulation and induces serious symptoms.

The patient's attention is first attracted by an increase in the size and firmness of his abdomen. In some instances icterus accompanies, or even precedes, this increase of girth. At length, driven to seek medical advice, he is discovered to have ascites usually without edema of the lower extremities. The physician examines the heart and urine, detects no heart disease and discovers no albumin, but perhaps some bile. The case is put down as one of hepatic cirrhosis. The following is an illustrative case:

Not long ago I saw in consultation a man of 55, who had been intensely jaundiced for nearly two years, and in August, 1900, was tapped for ascites. This had rather speedily recurred, and had been reduced by apocynum cannabinum for a time, but had again been drawn off the morning of the day I saw him. He had suffered from articular rheumatism eighteen years before, but experienced no shortness of breath or other discomfort since. The thin bordered, dense, slightly granular feeling liver extended in the median line nearly to the umbilicus and from one costal arch to the other, being lost beneath the ribs just outside the right mammillary line. Owing to the recent paracentesis, the peritoneal cavity was free from fluid and there was no edema.

The cardiac area was somewhat increased to the right and downward, the sounds were clear and strong and free from murmurs. The apex beat was rather tapping in character, in the 5th left interspace within the nipple line, and there was not very well marked epigastric pul-

sation. In the 5th and 6th interspaces between the apex beat and sternum, and also in the sulcus between the ensiform appendix and left costal cartilages, a systolic retraction could be perceived both by palpation and inspection. Furthermore, when the patient was instructed to draw a full slow breath, the right external jugular could be seen to bulge out during the inspiration. This distension was also palpable. Pulsus paradoxus could not be determined. I had no hesitation in making a diagnosis of pseudo-cirrhosis of the liver, secondary to adherent pericardium. Such cases may run a protracted course, requiring repeated tapplings, and the patients succumb to exhaustion, if not to the effects of stasis.

DIAGNOSIS.

Under some circumstances this may be one of the easiest of matters, in other cases it is one of the most difficult. Diagnosis is difficult, if not impossible, when the sac is adherent to the heart, but not to the neighboring structures. The signs then relied upon are, inspiratory distension, instead of normal inspiratory collapse of the external jugulars, or other superficial veins; diastolic collapse of the cervical veins, known as Friedreich's sign; pulsus paradoxus, a by no means constant or reliable sign; and the detection of cardiac hypertrophy for which no adequate cause can be discovered. When the pericardium is adherent to the chest wall or diaphragm, other signs are often developed that render diagnosis easy and certain. These are, a systolic retraction of one or more interspaces in close proximity to the apex beat or of the epigastrium; fixation of the apex, so that its gravitation from side to side with the turning of the patient's body or its descent during inspiration is prevented, or much restricted; a systolic retraction followed by a diastolic rebound of the chest wall that, by some authors, is considered pathognomonic; a systolic sinking or drawing in of the 10th and 11th intercostal spaces below the inferior angle of the left scapula, and occasionally of the right, known as Broadbent's sign. These are all very significant and much more frequently discovered than are those previously mentioned. In some instances auscultation detects friction sounds of a fine crackling or creaking character along the sides or apex of the sac, which sounds are synchronous with cardiac contractions and are sometimes elicited only during inspiration. A fine creaking sound is heard in some cases at the base of the heart upon the body of the sternum, while the patient slowly raises and lowers the arms. The detection of such pleuro-pericardial, or even strictly pleural friction sounds, furnish indirect or corroborative evidence of the existence of an adherent pericardium, and, taken in connection with cardiac hypertrophy and hepatic engorgement, would render the diagnosis extremely probable, even in the absence of more distinctive signs.

Finally, in some cases in which positive signs can not be obtained, the diagnosis of this condition is rendered possible by a process of exclusion, together with the history of a previous rheumatic attack, and the discovery of an hepatic enlargement for which no other predisposing cause can be ascertained. The elaboration of this subject as well as the prognosis and treatment will have to be left to the speakers that follow.

Isolation Hospital.—Colorado Springs, after a fruitless endeavor to combine with El Paso County in the erection of an isolation hospital, has decided to build one for the city alone, at a cost of \$2000.

TUBERCULAR PERICARDITIS.*

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AIKEN, S. C.

This is a disease much more prevalent than has heretofore been generally accepted. A great many cases of obscure heart trouble in the anemic when we find no valvular disease, and when there are no marked, but certain masked symptoms of the disease, is due to tubercular pericarditis. Especially is this true if the patient later begins to lose weight and assume a cachectic appearance.

Tubercular pericarditis generally progresses insidiously. It is communicated to the pericardium through the lymphatics, arterial or venous systems, and from that tends to extend to the peritoneum and other serous membranes. It is difficult to diagnose the disease as tubercular pericarditis in contradistinction to the non-tubercular pericarditis, unless it is secondary to a demonstrable tubercular lesion. However, if we are in doubt we can examine some of the aspirated fluid for the tubercle bacilli. If our search were negative we could not be positive that the disease was not tubercular, for the bacilli are very hard to find. It would then be necessary to inoculate a guinea-pig with the fluid, observing precaution of asepsis and if, after death, the glands are found to contain bacilli the diagnosis would be tubercular pericarditis.

The exudate is generally of the plastic, fibrinous, cheesy, or purulent form.

The symptoms of tubercular pericarditis are those that we would expect from an enlarged and adherent heart, together with the general symptoms of malaise and more disturbance of the general system than is found in the simple pericarditis, or that secondary to rheumatism or one of the exanthematous diseases.

CASE 1.—Male, white, aged 19, had been under my care for phthisis. The disease started in the apex of the right lung and progressed to stage of cavity, which was on the right side, behind second rib. Case was quiescent; the cavity was contracting; expectoration lessening rapidly, and he had stopped having an afternoon fever and night sweats.

The case was progressing satisfactory until the patient suddenly complained of pain in left side, became short of breath; his pulse rose to over 160; temperature became high and there was dullness over whole of left side. Upon careful examination the voice sounds were found and some râles at the apex of the left lung. The area of heart dullness was greatly enlarged and the sounds indistinct; the to-and-fro friction sounds, so significant of pericarditis, were audible. There was slight bulging of the intercostal spaces, but there did not seem to be enough to aspirate, and as the patient was tubercular it was not necessary to examine the fluid. The patient gradually became worse from the general tuberculosis and died from tuberculous diarrhea. The valves remained good throughout the illness and the brain was clear. In the last week only did the urine show marked albumin.

The interesting points in this case were the sudden involvement of the left lung and pericardium, when the case was apparently progressing favorably. At first it seemed possible that pneumonia was developing, and it was only after careful examination that pericarditis was diagnosed. There was not the large amount of fluid which we generally expect to find in the tubercular pericarditis, and it is to be regretted the more on that account that a postmortem was not permitted. Tubercle bacilli were found in the sputum.

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